MEDICAL PATHOLOGY AND THERAPEUTICS, AND PRACTICAL MEDICINE.

12. *Leukæmia a Primary Blood Disease.*—Prof. Biesiadecki, of Cracow, has lately advocated a new theory as to the nature of lukæmia, founded on the results of the careful anatomical and microscopical examination of certain cases which have come before him, as well as on other considerations. He believes leukæmia to be a primary disease of the solid elements of the blood (*eine Parenchymerkrankung des Blutes*), in which the white corpuscles are produced normally, but are afterwards prevented from developing into red corpuscles by retrograde changes which they undergo, so that the number of the latter cells appears to be diminished.

Biesiadecki regards the anatomical alterations in the spleen, lymphatic glands, liver, kidneys, etc., as due *secondarily* to the altered proportions of the elements of the blood. His reasons for this opinion are as follows (*Centralblatt*, No. 44, s. 757): 1. Neither the spleen nor the lymphatic glands, although so much increased in size, exhibit such changes as should lead one to conclude that there must be a greater production of white blood corpuscles in consequence. 2. In leukæmia, not only the parenchyma of the spleen, but also of the enlarged liver, and of the kidneys, is not only not hypertrophied, but, on the contrary, is in a state of atrophy. 3. The colourless blood corpuscles are not only relatively more numerous in leukæmia, but they are also of greater size, owing to a kind of colloid degeneration of their protoplasm. 4. These degenerated cells are deposited in the same organs and parts of organs as those in which blood cells containing pigment or vermilion collect. 5. In Biesiadecki's special case, a striking alteration was observed in the blood, before any swelling of the lymphatic glands or any sign of the tumours which afterwards appeared in the skin had been noticed. 6. After extirpation of the spleen in animals, neither the blood nor any organ exhibits important alterations.

While speaking of leukæmia, we may mention that Dr. Ordenstein, of Paris (*Centralblatt*, No. 42, s. 709), suggests the possibility of a connection between that disease and hereditary syphilis. He discovered that the father of a patient of his with splenic leukæmia had had constitutional syphilis, and this fact led him to try anti-syphilitic remedies on the son. Van Swieten's liquid—a solution of bichloride of mercury—was given for several months with surprising benefit, all other previous treatment having signally failed. The author promises to publish the case *in extenso*.—*Med. Times and Gaz* , Nov. 13, 1875.

13. *Pathological Anatomy of Endemic Malarial Fevers.*—In the *Archives de Physiologie*, No. 5, 1875, Prof. Kelsch, of the military hospital Val-de-Grâce, gives an account of a study of the changes found in the blood of patients suffering from malarial fever, made by means of this very valuable method.

The conclusions arrived at are, that in this fever there is—(1) A diminution in the number of the cellular elements of the blood ; (2) An augmentation in the size of the red globules; (3) A development of a black pigment which does not enter into the normal composition of the fluid. The observations extend over three classes of cases—simple fevers, pernicious (malignant) fevers, and cases of cachexia.

As an instance of the method of observation, the following case is translated entire:—

Obs. 1.—F., æt. 23, was 21 months in Africa. Had fever for the first time in the middle of August, 1874. Had three attacks of quotidian ague. On the 24th August, was admitted into the hospital for an organic affection of the heart. The fever relapsed in the hospital on 2d September ; paroxysm severe between 11 A. M. and 3 P. M. 3d September, morning, free from fever; red globules, 4,273,240 in a millimetre cube. 10 A. M., rigors followed by hot stage. 3 P. M., pulse 120; temperature 40.2° (Centig.); red globules 3,441,160. 4th September, morning, pulse 96; temperature 37°; red globules 3,209,724;

sulphate of quinine in frequent doses. There are no more paroxysms. Discharged the 25th. In twenty-four hours this patient lost more than a million of globules per millimetre cube.

In cases of prolonged fever, and in cachectic cases, the loss was much greater. Professor Kelsch states, *en résumé*, that when the variations in the number of globules in a vigorous man are followed from the first paroxysm to the stage of profound anæmia, it is found—(1) That the anæmia comes on very rapidly. In less than a month the globules fall from five millions to a million and a half or less per millimetre cube; (2) That there are three stages in the numerical variations of the red globules, which correspond with tolerable accuracy to the changes in the fever, as observed clinically. A first and short one, in which the globular deficit, exceedingly rapid, is reckoned by several hundred thousands daily. It corresponds to the initial stage of the fever, when there are acute febrile symptoms of more or less gravity, which are continued or remittent.

A second, which lasts longer than the first, during which the deficit for each paroxysm becomes less. The fever is still severe, but the intermitting type (quotidian or tertian) has taken the place of the remittent. During these two first stages the globules have continued to decrease in number.

Finally, a third, in which the number of globules ceases to fall. It oscillates at first for some time around a minimum. The blood still loses under the influence of the paroxysm, but the losses are less marked than in the two first periods, and are compensated by the salutary influence of periods free from fever, during which the globules are regenerated. This regeneration seldom takes place during the two preceding stages. Clinically, this last phase is marked by isolated attacks, between which there are longer or shorter periods, during which there is no fever.

Professor Kelsch further gives a number of measurements which show that the red globules increase in size, and that in malignant cases white corpuscles are observed, containing masses of pigment.—*Edinburgh Med. Journ.*, Dec. 1875.

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14. *The Treatment of Paludal Fevers by Arsenic.*—This paper, by M. SISTACH (*Archives Générales de Médecine*, September, 1875), comprises the fruits of a large experience gathered at the military hospital of Bona, in Algeria, one of the most malarious stations in that malarious colony. The author arranges his cases under four heads: intermittent faver, remittent fever, pernicious paludal fever, and paludal cachexia. The first group includes 229 cases, of which 136 were quotidian, seventy-four tertian, eleven quartan, five irregular, and three masked. Algerian intermittents are most frequent and severe during the summer months. The number of paroxysms is always greater in the first attack than in subsequent ones; and this difference is observed, whatever be the mode of treatment employed. Indeed, M. Laveran noticed it in his account of eighty-eight cases treated on the expectant method of Blidah. In comparing the results of treatment with those of simple expectation, it is indispensable that the cases should all have been subject to the same telluric and climatic conditions. In a comparatively healthy locality, such as Blidah, recovery without specific treatment is not uncommon; whereas at Bona, where the patient continues to be exposed to miasmatic influences of a most intense kind, attempts at expectation may prove dangerous, if not fatal. Each of the 136 cases of quotidian ague furnished an average number of 1.7 paroxysms after the treatment by arsenious acid had been begun; this average only amounting to 0.75 in the seventy-four cases of tertian fever. Frémy, Fuster, and Girbal had previously noticed that the febrifuge action of arsenic manifested itself more promptly in the latter than in the former variety of the disease.

The following are the rules laid down by the author for the administration of the remedy. They are essentially the same as those originally promulgated by Boudin; and the failures which have been recorded since his time by various observers must be attributed to their neglect of those details on which the certainty and the safety of arsenical medication depend. 1. The arsenious acid must be given in solution. The previous administration of an emetic is only